# Study on Pathogenicity of the *Aspergillus* species in experimentally immunosuppressed mice

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**Abstract**

*Aspergillus,* a saprophytic mould, distributed widely in the environment is a frequently recognized etiological agent in a number of disease conditions especially in immunocompromised patients. Mice untreated as well as immunocompromised by administering cortisone, were inoculated intraperitoneally with spores of *Aspergillus* spp. The administration of cortisone rendered mice highly susceptible to fatal visceral aspergillosis. Heat killed spores produced no evident lesions in control or experimental mice.

**Key words** : *Aspergillus*, Immunosuppression, Corticosteroid, mice.

## Introduction

Recent years have witnessed dramatic changes in man's environment and his immune defenses. Increasing incidence of secondary infections due to opportunistic fungi such as *Aspergillus* has been noted. Members of the genus *Aspergillus* are ubiquitous in nature and can survive under various conditions. *Aspergillus,* a conidia bearing fungus cause multiple diseases in human. These diseases include invasive aspergillosis, aspergilloma, different forms of hypersensitivity diseases, etc. The rising incidence of these infections in patients has been attributed to the widespread use of multiple antibacterial antibiotics, corticosteroids, irradiation, cytotoxic and other immunosuppressive drugs in clinical practice, increased incidence of acquired immunodeficiency syndrome (AIDS), autoimmune diseases and diabetes, coupled with stressful life styles. The literature abounds with reports of pathogenic potential of the *Aspergillus* species and the role of various predisposing factors on the susceptibility of host to these opportunistic fungal infections, demonstrated in experimental animals (Sidransky *et al.*, 1965, Ford and Friedman, 1967, Sandhu *et al.*, 1970, White, 1977, Thurston *et al.*, 1979, Hassan and Selim, 1983, Chattopadhyay *et al.*, 1994, Atasever *et al.*, 2004, etc.). But todate not much work has been done on the pathogenic potential of *Aspergillus* isolated from processed and ready to eat milk product. This study was undertaken to study the pathogenicity of a strain of *Aspergillus* isolated from *khoa* and to observe that whether cortisone would alter the susceptibility of mice to the pathogen after the intraperitoneal administration of spores of *Aspergillus* spp.

## Materials and Methods

*Aspergillus* spp. was isolated from a *khoa* sample procured from a retail shop in Mhow. The fungus was tested for its pathogenicity in the immunocompromised host. Spore suspension for inoculation was prepared by growing the organism on Potato dextrose agar medium at 22°C until profuse sporulation had occurred, usually in 4 to 6 days. The spores were harvested by addition of sterile normal saline with 0.1% of Tween 80 and shaking with glass beads. Tween 80 was added to spore suspension to avoid their clumping. Large particles were allowed to sediment under gravity and the supernatant spore suspension was decanted. After counting in a hematocytometer chamber a definite number of spores were used for inoculation.

Swiss albino mice weighing about 18-20g, bred in the small animal house of Institute of animal health and biological products, Mhow, were purchased for the study. Treated mice received 5 mg hydrocortisone, subcutaneously for 2 days before the spores were injected. 5 x $10^6$ number of spores were injected in mice *via* intraperitoneal route. Untreated control mice were injected with same number of spores *via* same route at the same time as the cortisone treated mice.

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Mice with and without treatment by cortisone were also inoculated with spores previously heated at 103°C for 24 hours. Their non-viability was confirmed by failure to grow on potato dextrose agar medium.

The rooms and cages used for housing the animals were thoroughly cleaned time to time. The animals were reared under strict hygienic conditions during and before infecting them. All the animals were adjudged to be healthy. The animals were provided with food and water *ad libitum*. The animals were observed daily for any morbidity or mortality. The mice died were necropsied within a short time.

**Result**

On intraperitoneal inoculation of spores of *Aspergillus* spp. in mice, the mortality was found in mice pretreated with hydrocortisone. Deaths occurred between three to ten days after exposure to spores of *Aspergillus* spp. Untreated or non - immunosuppressed mice were resistant to infection. On necropsy, lesions of visceral aspergillosis were observed. The parietal and serosal peritoneum appeared moist. Adhesions between visceral organs were found. Yellowish grey colour granulomas or abscesses were found in liver and kidneys. Heat killed spores produced no evident lesions in control or experimental mice.

**Discussion**

Mice are normally resistant to infection with *Aspergillus* and other saprophytic fungi. Hence, they are suitable animals for testing the possible role of cortisone drug in reducing resistance (Sidransky and Friedman,1959). In choosing the immunosuppressive regimen, corticosteroids were selected because of their profound effect on macrophage function, immobilization of phagocytes, stabilizing their lysosomes, diminished phagocytosis, low antibody production or impairment of antigen-antibody interaction in accordance with Louria and Brown (1960), Weissmann (1964) and Spreadbury *et al*. (1989).

The result of this study, which reveal that the administration of cortisone enhances the susceptibility of mice to the spores of *Aspergillus* spp. Being injected intraperitoneally, are consistent with earlier reports of Sidransky *et al*. (1972). Also, in conformity with his observations on *Aspergillus* sp., the fungal infection in the cortisone treated mice was found confined to the liver and kidney. Cortisone treatment would seem to impair the defenses, which prevent conidial germination, and also presumably those defenses that remove the conidia, which germinate. It therefore appears that host defenses exist in these organs and are inhibited by the cortisone treatment.

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